Response rate for food and water in the rat as a function of noncontingent, reinforcing septal stimulation¹

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Rats with electrodes in the septal region of the brain received noncontingent, reinforcing stimulation while bar pressing for food and for water on variable ratio schedules. Rate of pressing for water was found to be significantly reduced as a function of intracranial stimulation. No significant differences were found between stimulation and nonstimulation periods for Ss reinforced with food. The data revealed no change in general activity as a function of the electrical stimulation.

Is reinforcing septal stimulation intrinsically reinforcing, or are its reinforcing properties a result of activation of certain neural circuits concerned with organic drives such as hunger and thirst? Rate of bar pressing for septal stimulation is known to be a direct function of the length of deprivation for food and water (Brady, Boren, Conrad, & Sidman, 1957). Likewise, lesions in the septal area have been shown to influence food and water consumption (Harvey & Hunt, 1965; Harvey, Lints, Carlton, Jacobson, & Hunt, 1965; Pizzi & Lorens, 1967; Simmons & Thomas, 1961). When reinforcing septal stimulation is contingent upon eating or drinking, it tends to inhibit these consummatory activities (Asdourian, 1962; Robinson & Mischkin, 1962). Such inhibition could be predicted from a drive reduction concept of septal function (Miller, 1961), or from a reinforcement induction-drive induction concept (Deutsch & Howarth, 1963). A subsequent prediction might hold that noncontingent, reinforcing septal stimulation would result in a decrease in bar pressing for food and water. The present study was designed to test this prediction.

METHOD

Nine experimentally naive male albino rats served as Ss. The animals had a preoperative mean weight of 298 g and were approximately 150 days old at the beginning of experimental testing. Stereotaxic implantation of chronic electrodes into the septal region of the brain (0.75 mm anterior to bregma, 0.25 mm lateral to the midline, and 6.0 mm below the dura) was accomplished aseptically with the Ss under sodium pentobarbital anesthesia. Electrodes were bipolar, constructed of twisted strands of 0.1 mm diameter nicrome wire embedded in Amphenol strip connectors and insulated except for 0.25 mm at the tips. All electrode placements were verified histologically as being in the septum.

Pretesting for the reinforcing effect of septal stimulation was conducted in a gray 8 x 18 in. runway with 12 in. high walls. Leads to brain electrodes were suspended in such a way that movement about the runway was not restricted. Each S was allowed a 30 min adaptation period in the runway on the second day of water (Group 1) or food (Group 2) deprivation. Twenty-three hour deprivation was maintained for the eight consecutive days of pretesting. Beginning on the third day of deprivation, all Ss received intracranial stimulation at 2 sec intervals when at the end of the runway least preferred during adaptation. A Grass S4 stimulator provided a 0.5 sec train of biphasic square pulses of 50 microamperes (0.5 msec duration, 60 Hz). Stimulation parameters were constant throughout the experiment. During the first four days of pretesting, Ss were required to remain in the stimulation-contingent area of the

apparatus for 25 min out of one of the daily 30 min periods in order to meet criterion. When Ss had met criterion on one side of the apparatus, the procedure was reversed. Of 26 Ss, 10 (five in each reward group) met criterion at both ends of the apparatus and served as Ss for subsequent testing. (One S later became ill and was dropped from the experiment.)

Following completion of pretesting by both groups, deprivation schedules were reinstituted for two days before bar press training began. Training and testing were conducted in a transparent Plexiglas box (9 x 14 x 13 in.) with grid floor and a bar and food or water vendor at one end. Electrode leads were suspended from overhead such that they did not interfere with the Ss' movement about the cage. Group 1 (water deprived) received 0.05 ml of water for each bar press, and Group 2 (food deprived), a 45 mg rat chow pellet. When reliable performance levels were reached, all Ss were placed on a variable ratio schedule (mean ratio = 8:1), and training was continued until a stable pressing rate had been maintained for at least three days. Ss were then placed in the cage for a 30 min experimental session on each of four consecutive days, during which food (Group 1) or water (Group 2) was available under the same variable ratio schedule employed in training.

Each experimental session was subdivided into six 5 min periods ($P_1 - P_6$). Three of these periods served as treatment periods in which Ss received intracranial stimulation noncontingently on a variable interval schedule with a mean interstimulus interval of 40 sec (range: 10 sec to 90 sec). The remaining three periods served as control periods during which no stimulation was delivered. Treatment periods and control periods were alternated within days and between days in order to control for any periods effect.

General activity of Ss during testing was monitored by (1) six photocell units directed across the width of the box at equal intervals, three units 2 in. above the floor and three units 6 in. above the floor, and (2) two units directed across the length of the box from either side of the bar. Photocell responses were indicated by pen deflections on an eight channel polygraph.

RESULTS

Because the effects of intracranial stimulation on rate of bar pressing were not expected to be long-lasting (Deutsch & Howarth, 1963), response rate during the 10 sec intervals immediately following each stimulation was selected as data for the analyses. Control data consisted of the rate of responding during corresponding intervals in the control periods. In order to detect any change in general activity as a function of the stimulation, the number of photo beam interruptions occurring during the same 10 sec response intervals was also analyzed.

As predicted, the mean rate of responding for water (Group 1) was significantly less during the noncontingent septal stimulation periods than during the control (nonstimulation) periods ($t = 3.60$, $p < .025$, $df = 4$, one-tailed). Extrapolated to the usual hourly basis, mean rates of bar pressing were 2952 and 3024 for stimulation and nonstimulation periods, respectively.

The effect of septal stimulation upon responding for food, although resulting in fewer bar presses during stimulation than control periods, was not statistically significant ($t = 1.82$, $df = 3$). In terms of presses per hour, the mean rates were 2232 and 2268 bar presses for the stimulation and nonstimulation intervals, respectively.

Activity scores for Groups 1 and 2 indicated no effect of

septal stimulation upon general activity, as monitored by the photocell units, although there were significant day to day fluctuations in activity in Group I (water deprived) independent of intracranial stimulation. No evidence of distraction or motor interference as a result of the intracranial stimulation was found in polygraph records or observation for either group. Normal activity patterns were uninterrupted during and following stimulation. At no time during or following stimulation were the Ss observed to withdraw from the bar, freeze, shudder, or succumb to motor seizure.

DISCUSSION

The results of this study are consistent with the hypothesis that the presentation of noncontingent, reinforcing septal stimulation can decrease the frequency of behavior maintained by reinforcement with water. The difference in bar pressing between treatment and control conditions is not great; however, this was most likely due to widely spaced stimulations of short duration.

One possible explanation for the decrease in rate of bar pressing is that the reinforcing intracranial stimulation reduces the effectiveness of water as a reinforcer. Whether this might result from electrical activation of some inhibitory circuit and consequently "drive reduction" (Miller, 1961), or from activation of "reinforcement pathways" (Deutsch & Deutsch, 1966) concerned with thirst is not clear. It should be noted that the latter interpretation postulates a lower threshold for drive than for reinforcement pathways. Using Deutsch's criteria (Deutsch, Howarth, Ball, & Deutsch, 1962), septal stimulation in the present study was obviously at or above threshold for both thirst drive and thirst reinforcement systems. In this context the significant decrease in rate of bar pressing for water would seem to indicate that even though the thirst reinforcement system has the higher threshold, it predominated over (i.e., excluded) the effects of thirst drive system stimulation. It might have been predicted that there would be decreased bar pressing with noncontingent intracranial stimulation (Deutsch et al. 1962) because of the reinforcement of competing responses. However, Ss in the present study obviously preferred water to septal stimulation. They spent virtually all their time at the bar and water dispenser; no instances of amplification of new responses or establishment of "cyclic superstitious sequences of behavior" occurred.

Explanations of these results in terms of electrical interference and blocking of neural activity in general would seem to be precluded by the relatively restricted neural field activated by such stimulation (Valenstein, 1966). The possibility remains that the behavioral changes obtained are simply manifestations of central alteration of sensory input, but this in itself may be considered a drive mechanism (Valenstein, 1966).

The failure to find a significant effect of septal stimulation on bar pressing by food deprived Ss may indicate differentiation of reinforcement systems for food and water, although similar trends in the scores for the two groups and the well-established functional relationships between food and water deprivation (Cizek & Nocenti, 1965; Jacobs, 1964) confirm the interdependence of hunger and thirst.

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NOTES

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